

Transient atrial fibrillation after minor head injury

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Two patients developed transient rapid atrial fibrillation after a blow on the head. There was no evidence of neurological damage or organic heart disease on subsequent investigation. Neither patient was aware of the cardiac irregularity.

Atrial fibrillation most commonly results from organic heart disease but may occur in patients with clinically normal hearts (Friedlander and Levine, 1934). Sometimes in these cases it follows stressful insults and has been recorded after pain, a period of heavy smoking, or excessive drinking of alcohol. Cranial trauma appears to have induced the arrhythmia but in the few previous reports the injury was severe and intracranial haemorrhage sustained (Marks, 1956; Bonofiglio, Bugaro, and Pantaleoni, 1967; Palma *et al.*, 1971). The association of atrial fibrillation after trivial head injury has not been previously clearly recognized.

Case reports

Case 1

A fit 19-year-old man was being examined for entry into the forces. His heart was found to be normal. When the medical officer tried to look into his left ear with an auriscope he fell over and struck his head on a cupboard. He lost consciousness for about a minute and his pulse was rapid and irregular. He was sent to hospital where an electrocardiogram confirmed atrial fibrillation with a few ventricular ectopic beats. The patient at no time felt palpitation and was otherwise well. A chest x-ray film was normal. No treatment was given. On review, a week later, clinical examination of his heart and the electrocardiogram showed no abnormality.

Case 2

A 32-year-old heavy labourer was admitted to hospital after a fight during which he had fallen over, struck his right forehead, and lost consciousness for a short time. On the evening of admission he had drunk 4 pints of beer. He had had no pre-

vious illness and was not a smoker. On examination there were no abnormal neurological signs but he had a fast and irregular heart beat. An electrocardiogram showed atrial fibrillation at a rate of 150 beats a minute. He was unaware of the arrhythmia. Twenty-four hours later he had reverted to sinus rhythm and his heart was normal on clinical examination. An electrocardiogram recorded at that time showed no abnormality. A chest x-ray film was normal and serum thyroxine was 114 nmol/l (normal range 70 to 160 nmol/l). The patient made an uneventful recovery.

Discussion

Both patients developed rapid atrial fibrillation after a blow to the head. Neither had primary cardiac disease. The fact that the development of the abnormal rhythm was noted and recorded directly after the cranial injury suggests that these events were related.

It is known that cerebral lesions may influence cardiac action. Electrocardiographic changes sometimes follow intracranial haemorrhage (Menon, 1964) and transient atrial fibrillation followed subarachnoid haemorrhage in 1 of 12 cases of this condition reported by Shuster (1960). The mechanism may be a reduction in the atrial refractory period mediated by vagal stimulation, as atrial premature beats can induce atrial fibrillation (Killip and Gault, 1965). This seems particularly likely to occur where simultaneous sympathetic overactivity also increases the heart rate. The blow on the skull in these patients may have induced such vagal and sympathetic excitation. In the first patient it is also possible that the manipulation of the auriscope in the external auditory meatus was the initiating factor. Fowler and Baldrige (1931) reported atrial fibrillation in a previously fit 24-

year-old medical student who fainted after removal of ear wax.

It is interesting that neither patient was aware of the rapid irregularity of the heart as this is usual in paroxysmal atrial fibrillation. It is possible that transient arrhythmias may not be uncommon after head injury.

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